L6 ANSWER 47 OF 55 CANCERLIT
ACCESSION NUMBER: 97149734 CANCERLIT
DOCUMENT NUMBER: 97149734 PubMed ID: 8996528
TITLE: DNA damage inducible-gene expression following platinum treatment in human ovarian carcinoma cell lines.
AUTHOR: Delmastro D A; Li J; Vaisman A; Solle M; Chaney S G
CORPORATE SOURCE: Department of Medicine, School of Medicine, University of North Carolina, Chapel Hill 27599, USA.
CONTRACT NUMBER: 5-T32-H107149-19 (NCI)
CA34082
SOURCE: CANCER CHEMOTHERAPY AND PHARMACOLOGY, **(1997)** 39 (3) 245-53.
Journal code: 7806519. ISSN: 0344-5704.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: MEDLINE; Priority Journals
OTHER SOURCE: MEDLINE 97149734
ENTRY MONTH: 199702
ENTRY DATE: Entered STN: 19970305
Last Updated on STN: 19970509

AB PURPOSE: DNA damage-inducible genes, such as gadd153, gadd45, **p21** and c-jun, have previously been shown to be induced by the chemotherapeutic agent cisplatin. One of these genes, gadd153, has previously been reported to be differentially expressed in cisplatin-resistant cell lines and, therefore, to be a potential prognostic indicator for tumor response to cisplatin-based chemotherapy. It is not currently known whether such damage-inducible genes are turned on by the DNA damage itself (e.g. by the formation of Pt-DNA adducts) or by the downstream biological consequences of that damage. It is also not known whether the increased expression of these DNA-damage-inducible genes is related to immediate protective responses such as DNA repair or to more delayed responses such as cell cycle arrest or **apoptosis**. These experiments were initiated to characterize more fully the nature of the DNA damage-inducible **response** to cisplatin **treatment** and to determine whether any of these genes might be useful prognostic indicators of tumor response to cisplatin chemotherapy. METHODS: The dose-response and time-course for the induction of the DNA damage-inducible genes gadd153, gadd45, **p21** and c-jun were examined by Northern analysis in the human ovarian carcinoma cell line 2008 and its resistant subclone C13* following treatment with platinum anticancer agents. The extent of gene expression was correlated with cytotoxicity determined by growth inhibition assay, Pt-DNA adducts determined by atomic absorption spectrometry and inhibition of DNA synthesis determined by 3H-thymidine incorporation. RESULTS: All four genes were induced maximally in both sensitive and resistant cell lines at lethal cisplatin doses (> or = ID90). Induction was maximal between 24 and 48 h following exposure to the drug for all genes except c-jun which was induced by 6 h. At 24 h following cisplatin treatment the overall levels of gadd153 were less in the resistant C13* cell line than in the parental 2008 cell line, while those of gadd45 were greater in C13* than in 2008. Maximal expression of **p21** and c-jun was not significantly different in the two cell lines. The dose-response of these genes

correlated with the cytotoxicity of cisplatin and the inhibition of DNA synthesis by cisplatin, rather than to the actual levels of Pt-DNA adducts. The more cytotoxic platinum analog, ormaplatin, also induced gadd153 and its induction was also based on cytotoxicity. CONCLUSION: These results suggest that the regulation of gadd153 and gadd45 expression occurs thorough separate pathways in the 2008 and C13* cell lines. The DNA damage-inducible gene response for all four damage-inducible genes tested appeared to be more directly correlated with downstream biologic effects of cisplatin damage than with actual Pt-DNA adduct levels. The time-course and dose-response for induction of these genes was more consistent with delayed responses such as **apoptosis** rather than more immediate responses such as DNA repair. Finally, these results strengthen previous suggestions that the expression of gadd153, and possibly other DNA damage-inducible genes, may be useful indicators of tumor response to cisplatin-based chemotherapy.

L6 ANSWER 49 OF 55 CANCERLIT
ACCESSION NUMBER: 1998642022 CANCERLIT
DOCUMENT NUMBER: 98642022
TITLE: Hematopoietic and cytogenetic responses to novel anti-cytokine therapy in myelodysplastic syndromes (MDS) (Meeting abstract).
AUTHOR: Raza A; Gezer S; Venugopal P; Kaizer H; Hines C; Thomas R; Alvi S; Mundle S; Shetty V; Borok R; Loew J; Reza S; Robin E L; Rifkin S D; Alston D; Hernandez B; Shah R; Hsu W T; Dar S; Gregory S A
CORPORATE SOURCE: Rush Cancer Institute, Chicago, IL.
SOURCE: Proc Annu Meet Am Soc Clin Oncol, **(1997)** 16 A22.
ISSN: 0732-183X.
DOCUMENT TYPE: (MEETING ABSTRACTS)
LANGUAGE: English
FILE SEGMENT: Institute for Cell and Developmental Biology
ENTRY MONTH: 199806
ENTRY DATE: Entered STN: 19980610
Last Updated on STN: 19980610

AB MDS are clonal stem cell disorders where the clinical paradox of pancytopenia despite cellular marrows has been ascribed to the presence of excessive cytokine-driven **apoptotic** death of hematopoietic cells (Raza, Blood; 86:268 1995). Three cytokines associated with the dual role of stimulating CD34+ progenitors to proliferate (hypercellular marrow) and inducing programmed cell death in their maturing daughters (pancytopenia) were found in excessive amounts in the majority of MDS patients namely TNF-a, **TGF-b** and IL1-b. Suppression of these cytokines was attempted by using pentoxifylline 800 mg tid, ciprofloxacin 500 mg bid and decadron 4.0 mg q am (PCD). Of 51 total MDS patients treated to date, 18 have responded with 5 patients also showing cytogenetic responses. In two successive protocols, 11 patients with RA, 1 with RARS, 4 with RAEB, 1 with RAEB-t and 1 with CMMoL **responded**. The **therapy** was successful in reducing rate of **apoptosis** and TNF-a/ **TGF-b** levels. The exact significance of cytogenetic responses is unclear without longer follow-up. These data demonstrate the validity of our thesis implicating the cytokines in the genesis of the clinical syndrome and represent a novel area for further therapeutic research.
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L6 ANSWER 50 OF 55 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE

L6 ANSWER 51 OF 55 CANCERLIT DUPLICATE 31
ACCESSION NUMBER: 1999035186 CANCERLIT
DOCUMENT NUMBER: 99035186 PubMed ID: 9816338
TITLE: Prognostic value of p21(WAF1) and p53 expression in breast carcinoma: an immunohistochemical study in 261 patients with long-term follow-up.
AUTHOR: Caffo O; Doglioni C; Veronese S; Bonzanini M; Marchetti A; Buttitta F; Fina P; Leek R; Morelli L; Palma P D; Harris A L; Barbareschi M
CORPORATE SOURCE: Departments of Histopathology, S. Chiara Hospital, Largo Medaglie d'Oro, 38100, Trento, Italy.
SOURCE: CLINICAL CANCER RESEARCH, **(1996 Sep)** 2 (9) 1591-9.
Journal code: 9502500. ISSN: 1078-0432.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: MEDLINE; Priority Journals
OTHER SOURCE: MEDLINE 1999035186
ENTRY MONTH: 199902
ENTRY DATE: Entered STN: 19990405
Last Updated on STN: 19990405

AB **p21** protein (**p21**) inhibitor of cyclin-dependent kinases is a critical downstream effector in the p53-specific pathway of growth control and can also be induced by p53-independent pathways in relation to terminal differentiation. We investigated **p21** immunoreactivity in 261 breast carcinomas (141 node negative and 120 node positive) with long-term follow-up (median, 73 months; range, 37-119). **p21** was seen in 214 (82%) infiltrating tumors, staining was nuclear and heterogeneous, and the **p21** labeling index ranged from 0 to 90%. Sixty-eight (32%) patients showed **p21** overexpression (>10% of reactive tumor cells). **p21** overexpression was associated with large tumor size, positive nodal status, high histological grade, and high mitotic count and was related to short disease-free survival (DFS) in the whole series of patients (P = 0.04), in the node-negative subgroup (P = 0.004), and in the group of patients who did not undergo systemic adjuvant therapy (P = 0.003). In patients treated with systemic adjuvant therapy, bivariate analysis of the combined **p21** and p53 phenotypes showed that **p21**+/p53+ tumors were associated with long DFS and overall survival (OS), whereas **p21**-/p53+ tumors had the worst prognosis. In treated patients, multivariate analysis showed that the **p21**-/53+ phenotype was independently associated with short DFS and OS. Our present data support the hypothesis that **p21**/p53 heterogeneous expression may be of clinical relevance for the **therapeutic response** to chemotherapy/hormonotherapy. The **p21**-/p53+ phenotype could correspond to a situation where p53 overexpression really reflects complete abrogation of p53 function. These cases with disrupted p53 function should have impaired the G1 checkpoint and may not be able to activate the **apoptotic** cascade in response to DNA-damaging drugs.

L6 ANSWER 52 OF 55 CANCERLIT
ACCESSION NUMBER: 96625785 CANCERLIT
DOCUMENT NUMBER: 96625785
TITLE: Factors involved in determining whether transforming growth factor beta suppresses the transformed phenotype and/or induces apoptosis (Meeting abstract).
AUTHOR: Reeder M K; Isom H C
CORPORATE SOURCE: Penn State Coll. of Med., Hershey, PA.
SOURCE: Proc Annu Meet Am Assoc Cancer Res, **(1996)** 37 A195.
ISSN: 0197-016X.
DOCUMENT TYPE: (MEETING ABSTRACTS)
LANGUAGE: English
FILE SEGMENT: Institute for Cell and Developmental Biology
ENTRY MONTH: 199606
ENTRY DATE: Entered STN: 19970509
Last Updated on STN: 19970509

AB We have developed a series of immortalized cell lines (the CWSV cell lines) by transfecting primary rat hepatocytes with SV40 DNA. Three cell lines, which differ in their status of tumorigenic progression, **respond** differently when **treated** with transforming growth factor beta (**TGFb**). An immortalized cell line, CWSV1, is essentially unaffected by **TGFb**. A weakly tumorigenic cell line, 14MP, is induced to undergo **apoptosis** by **TGFb**. A malignant tumor-derived cell line, 14T1, is suppressed in the transformed phenotype by **TGFb**. The purpose of this study was to identify factors that may contribute to these varied responses to **TGFb**. First, the expression of **TGFb** receptors was compared by crosslinking with 125I conjugated-**TGFb**. Using both cold **TGFb** competition and 125I-**TGFb** saturation assays, we found that the ratio of **TGFb** Type I to Type II receptors on the 14MP cells was much higher than the CWSV1 cells. Second, we used radiolabeling and immunoprecipitation to examine the three cell lines with and without **TGFb** treatment for p53 and SV40 T Antigen (TAg) expression and phosphorylation. Both p53 and TAg were essentially unaffected in 14MP cells. Synthesis and consequently, the level of phosphorylated p53 increased at late time points of **TGFb** treatment of 14T1 cells. The conclusions are (1) the varied responses of the cells to **TGFb** may be partially caused by differences in the **TGFb** receptor ratios, (2) **TGFb**-induced **apoptosis** in 14MP cells is not due to an alteration in p53 or TAg, (3) suppression of the transformed phenotype of 14T1 cells is not due to a decrease of TAg synthesis or phosphorylation and (4) an increase in the level and/or phosphorylation of p53 in 14T1 cells may play a role in suppression of transformed phenotype.

L6 ANSWER 54 OF 55 CANCERLIT
ACCESSION NUMBER: 96600846 CANCERLIT
DOCUMENT NUMBER: 96600846
TITLE: Restoration of apoptosis in p53 deficient tumor cells (Meeting abstract).
AUTHOR: Fisher D E; Bodis S; Lowe S; Takemoto C; Housman D; Jacks T
CORPORATE SOURCE: Div. of Pediatric Oncology, Dana-Farber Cancer Inst., Harvard Medical School, Boston, MA.
SOURCE: Blood, **(1994)** 84 (10, Suppl 1) 111a.
ISSN: 0903-1936.
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Institute for Cell and Developmental Biology
ENTRY MONTH: 199603
ENTRY DATE: Entered STN: 19970509
Last Updated on STN: 19970509

AB Successful killing of cancer cells, particularly hematologic malignancies, is increasingly recognized to occur through the induction of **apoptosis**, a cell-encoded suicide pathway modulated by factors such as p53 and bcl-2. p53 aberrations are the commonest gene defect recognized in human cancer and may confer refractoriness to **apoptosis** induction. Loss of **apoptosis** may potentiate propagation of malignant clones and simultaneously render such cells resistant to killing by many anticancer therapies. We have examined E1A/Ras transformed fibroblasts derived from p53 knockout mice or their p53 wild-type counterparts for **apoptosis** induction by chemotherapies and radiation in vitro and in a nude mouse tumor model. For virtually all treatments tested, including radiation, interchelators, antimetabolites, antibiotics, topo inhibitors, and alkylating agents, p53+ tumor cells underwent rapid **apoptosis** induction while p53- tumor cells were refractory. However Taxol, a microtubule targeting drug, killed tumor cells independently of p53 status and produced dramatic tumor shrinkage in nude mice. Nontransformed parental fibroblasts appeared relatively refractory to Taxol, potentially explaining tumor cell selectivity as also often occurs for p53-dependent **apoptosis**. DNA ladders, DAPI staining, and terminal transferase-nucleotide (TUNEL) staining revealed Taxol to kill via **apoptosis** in both p53+ and p53- tumor cells. The cyclin inhibitor **p21**/Waf/Cip is a known downstream target of p53. Recent data have suggested that **p21** may be induced independently of p53. Using northern blot analysis, we found that **p21** RNA expression was significantly induced by Taxol treatment in p53- tumor cells, suggesting that microtubule-mediated events may feed into cell cycle control pathways which regulate **apoptosis** induction. These studies demonstrate that **apoptosis** induction in vitro correlates with tumor **response** to antineoplastic **therapies** in an animal model. Antineoplastic therapies commonly used in humans required wildtype p53 to induce **apoptosis** and significant shrinkage. Taxol, which targets microtubules and functions independently of DNA damage, potently induced **apoptosis** in p53- tumors. The cyclin inhibitor **p21** was found to be induced by Taxol treatment and may provide a link to **apoptosis** induction

and sensitivity to anticancer therapy.

L31 ANSWER 8 OF 10 CANCERLIT
ACCESSION NUMBER: 1999285173 CANCERLIT
DOCUMENT NUMBER: 99285173 PubMed ID: 10356685
TITLE: Update on the management of advanced breast cancer.
AUTHOR: Fornier M; Munster P; Seidman A D
CORPORATE SOURCE: Breast Cancer Medicine Service, Memorial Sloan-Kettering Cancer Center, New York, New York, USA.
SOURCE: ONCOLOGY, **(1999 May)** 13 (5) 647-58; discussion 660, 663-4. Ref: 88
Journal code: 8712059. ISSN: 0890-9091.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: MEDLINE; Priority Journals
OTHER SOURCE: MEDLINE 1999285173
ENTRY MONTH: 199907
ENTRY DATE: Entered STN: 19990813
Last Updated on STN: 19990813

AB Recent trials comparing single-agent vs **combination** therapy in metastatic breast cancer suggest that it may be time to reconsider the belief that **combination** chemotherapy is the gold standard of treatment. Based on the limited randomized trial data available to date, high-dose chemotherapy with stem-cell rescue should not be viewed as "state-of-the art" treatment for metastatic disease and should be used only in the context of clinical trials. Recent trials have explored the optimal dosing and scheduling of the taxanes, as well as the possible role of these agents in **combination** regimens. Capecitabine (Xeloda), a new oral fluoropyrimidine, appears to be comparable in efficacy to CMF (cyclophosphamide, methotrexate, and fluorouracil), and preclinical data suggest possible synergy between this agent and the taxanes. Other promising agents under study include liposome-encapsulated **doxorubicin** (TLCD-99), an immunoconjugate linking a chimeric human/mouse monoclonal antibody to **doxorubicin** molecules; MTA (LY231514), a multitargeted antifolate; and marimistat, a broad-spectrum matrix metalloproteinase inhibitor. Tamoxifen (Nolvadex) remains the most important hormonal agent, but new antiestrogens and selective estrogen receptor modulators (SERMs) may provide alternatives. The potential role of new aromatase inhibitors as first-line hormonal agents requires further study. Finally, the possible synergy between trastuzumab (**Herceptin**), a recombinant humanized monoclonal antibody to the HER-2/neu protein, and paclitaxel (**Taxol**) is being studied in two clinical trials.

L31 ANSWER 9 OF 10 CANCERLIT
ACCESSION NUMBER: 2000140974 CANCERLIT
DOCUMENT NUMBER: 20140974 PubMed ID: 10676565
TITLE: New developments in chemotherapy of advanced breast cancer.
AUTHOR: Lebwohl D E; Canetta R
CORPORATE SOURCE: Bristol-Myers Squibb Pharmaceutical Research Institute, Wallingford, CT, USA.
SOURCE: ANNALS OF ONCOLOGY, **(1999)** 10 Suppl 6 139-46.
Ref: 64
Journal code: 9007735. ISSN: 0923-7534.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: MEDLINE; Priority Journals
OTHER SOURCE: MEDLINE 2000140974
ENTRY MONTH: 200003
ENTRY DATE: Entered STN: 20000413
Last Updated on STN: 20020726

AB Anthracyclines and taxanes are the two most active classes of chemotherapy for the treatment of advanced breast cancer. Recent studies have investigated **combination** therapy including **doxorubicin** (Dox) and paclitaxel. The efficacy of this **combination** has been established in a phase III study conducted by ECOG, comparing Dox/paclitaxel versus Dox versus paclitaxel. The **combination** is superior to Dox or paclitaxel with respect to response rate and time to disease progression, indicating that the **combination** provides a new standard for the first line treatment of metastatic breast cancer [1].

Phase II studies using higher doses of Dox and using shorter infusions of paclitaxel have suggested the **combination** can be further optimized; Gianni reported a 94% objective response rate using Dox 60 mg/m2 followed by paclitaxel 175 mg/m2 given over three hours [2]. The more active regimens are associated with enhanced cardiotoxicity; this toxicity can be avoided, however, by limiting the exposure to **doxorubicin**. The newer regimens have now been moved into phase III studies. Future progress for this disease will depend on the introduction of new agents. Two novel drugs are currently being investigated in randomised phase III trials as potentiators of Dox and/or paclitaxel. One is a monoclonal antibody from Genentech (**Herceptin**, trastuzumab) directed at the HER-2/neu oncogene, which is overexpressed in > 25% of breast cancers [3]. Recent results indicate that **Herceptin** in **combination** with paclitaxel (or with a Dox plus cyclophosphamide regimen) induces a higher response rate (RR) and prolongs the time to disease progression when compared to chemotherapy alone. The second agent N,N-diethyl-2[4-(phenylmethyl)-phenoxy] ethanamine.HCl (DPPE, BMS-217380-01), when **combined** with Dox, was associated with a higher RR than previously observed with Dox alone [4]. A randomized trial of Dox versus Dox plus DPPE is ongoing. The possible mechanisms underlying chemo-potentiation by these agents are discussed. As new anthracycline/taxane **combinations** establish themselves in earlier stages of the disease, the need for effective, non-cross resistant

salvage regimens will emerge.

L10 ANSWER 67 OF 77 CANCERLIT
ACCESSION NUMBER: 95609259 CANCERLIT
DOCUMENT NUMBER: 95609259
TITLE: **Markers** for differentiation and **apoptosis** as intermediate endpoints for the development of lung **cancer** (Meeting abstract).
AUTHOR: Zhang H; Yousem S A; Elder E; Whiteside T; Levitt M L
CORPORATE SOURCE: Medical College of Pennsylvania-Allegheny Campus, Pittsburgh, PA 15212.
SOURCE: Proc Annu Meet Am Assoc Cancer Res, **(1995)** 36 A1482.
ISSN: 0197-016X.
DOCUMENT TYPE: (MEETING ABSTRACTS)
LANGUAGE: English
FILE SEGMENT: Institute for Cell and Developmental Biology
ENTRY MONTH: 199508
ENTRY DATE: Entered STN: 19950809
Last Updated on STN: 19950809

AB In order to plan rational chemoprevention strategies for lung **cancer** we are attempting to identify intermediate endpoints for the development of **neoplasm** based on markers for squamous differentiation and apoptosis. Using immunohistochemical methodology we investigated the expression of proteins for both tissue and keratinocyte transglutaminases (tTG and kTG), involucrin, loricrin and Bcl-2, in both non-small cell lung **cancers** and nonmalignant lung tissues (although most of the latter were obtained from patients with lung **cancer**). While tTG was expressed in almost all samples, its distribution was markedly different in **tumor** when compared to nonmalignant tissue. Squamous **carcinomas** alone were kTG positive while co-expressing tTG; however, these two enzymes were differentially distributed. Almost all **tumor** samples expressed both involucrin and loricrin in a patchy distribution that was most prominent in squamous **carcinomas**. Some nonmalignant specimens also expressed these molecules, but very weakly. Bcl-2 oncoprotein was expressed in 74% of **tumors**, with weak or absent expression in nonmalignant specimens. Interestingly, areas expressing Bcl-2 vs tTG or kTG were mutually exclusive. In summary, markers for differentiation and apoptosis demonstrate potential for predicting the development of lung **cancer**. Differential expression may be even more marked when **tumors** are compared to tissues from healthy subjects. These markers may form a basis for the development of chemoprevention strategies for this disease.

L18 ANSWER 47 OF 59 CANCERLIT
ACCESSION NUMBER: 95613177 CANCERLIT
DOCUMENT NUMBER: 95613177
TITLE: Flow cytometric measurement of apoptosis in **leukemic** cells identified by a membrane antigen (Meeting abstract).
AUTHOR: de la Puerta M; Benson N; Scott M; Lynch J; Braylan R
CORPORATE SOURCE: Dept. of Pathology, Univ. of Florida, Gainesville, FL.
SOURCE: Proc Annu Meet Am Soc Clin Oncol, **(1995)** 14 A21.
ISSN: 0732-183X.
DOCUMENT TYPE: (MEETING ABSTRACTS)
LANGUAGE: English
FILE SEGMENT: Institute for Cell and Developmental Biology
ENTRY MONTH: 199509
ENTRY DATE: Entered STN: 19950906
Last Updated on STN: 19970509

AB Since apoptotic cell death has been implicated in the response of **leukemia** to therapy, the accurate quantitation of **leukemic** cells undergoing apoptosis may have prognostic significance. This quantitation may be particularly important in cases with resistant clones.

However, it would be difficult to accurately enumerate apoptotic **leukemic** cells in heterogeneous samples where the **neoplastic** cells represent only a fraction of all cells. We present here a flow cytometric method to recognize apoptotic **leukemic** cells in normal peripheral blood. We synthesized a **leukemic** model by mixing CD33-expressing HL-60 cells (a promyelocytic cell line) in equal proportions with ficoll hypaque-separated, monocyte-depleted peripheral blood lymphocytes from a normal donor. Apoptosis was induced by exposing the cell mixture to 10,000 uJ of ultraviolet (UV) radiation in a DNA cross-linking apparatus. Following an incubation period of 4 hr, DNA was extracted from a portion of the sample and analyzed on a 1% agarose electrophoresis gel for the presence of a 'ladder' to confirm induction of apoptosis. The remaining cells were exposed to phycoerythrin-conjugated, anti-CD33 monoclonal **antibody** or normal mouse IgG (negative control). The samples were then fixed with FACSLYSE [Becton Dickinson Immunocytometry Systems (BDIS)] and exposed to biotinylated dUTP in presence of terminal deoxynucleotidyl transferase (Tdt), followed by fluorescein isothiocyanate (FITC)-conjugated avidin to selectively label apoptotic cells. Cells not exposed to UV radiation, and cells exposed to UV and dUTP but in the absence of Tdt served as controls. All samples were analyzed on a FACSort flow cytometer (BD). Only the samples exposed to UV and dUTP in the presence of Tdt showed a subpopulation of FITC-labeled HL60 cells (identified by their light scatter properties and the expression of CD33). The normal lymphocytes in the mixture were not labeled by FITC. These results demonstrate the potential of using flow cytometric cell surface **marker** analysis to selectively measure **apoptotic** **leukemic** cells in heterogenous samples.
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L31 ANSWER 8 OF 10 CANCERLIT
ACCESSION NUMBER: 1999285173 CANCERLIT
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CORPORATE SOURCE: Breast Cancer Medicine Service, Memorial Sloan-Kettering Cancer Center, New York, New York, USA.
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General Review; (REVIEW)
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LANGUAGE: English
FILE SEGMENT: MEDLINE; Priority Journals
OTHER SOURCE: MEDLINE 1999285173
ENTRY MONTH: 199907
ENTRY DATE: Entered STN: 19990813
Last Updated on STN: 19990813

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PUB. COUNTRY: Netherlands
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LANGUAGE: English
FILE SEGMENT: MEDLINE; Priority Journals
OTHER SOURCE: MEDLINE 2000140974
ENTRY MONTH: 200003
ENTRY DATE: Entered STN: 20000413
Last Updated on STN: 20020726

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